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Neural control of interlimb oscillations

II. Biped and quadruped gaits and bifurcations

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Abstract. Behavioral data concerning animal and human gaits and gait transitions are simulated as emergent properties of a central pattern generator (CPG) model. The CPG model is a version of the Ellias-Grossberg oscillator. Its neurons obey Hodgkin-Huxley type equations whose excitatory signals operate on a faster time scale than their inhibitory signals in a recurrent on-center off-surround anatomy. A descending command or GO signal activates the gaits and triggers gait transitions as its amplitude increases. A single model CPG can generate both in-phase and anti-phase oscillations at different GO amplitudes. Phase transitions from either in-phase to anti-phase oscillations or from anti-phase to in-phase oscillations can occur in different parameter ranges, as the GO signal increases. Quadruped vertebrate gaits, including the amble, the walk, all three pairwise gaits (trot, pace, and gallop), and the pronk are simulated using this property. Rapid gait transitions are simulated in the order – walk, trot, pace, and gallop – that occurs in the cat, along with the observed increase in oscillation frequency. Precise control of quadruped gait switching uses GO-dependent modulation of inhibitory interactions, which generates a different functional anatomy at different arousal levels. The primary human gaits (the walk and the run) and elephant gaits (the amble and the walk) are simulated, without modulation, by oscillations with the same phase relationships but different waveform shapes at different GO signal levels, much as the duty cycles of the feet are longer in the walk than in the run. Relevant neural data from spinal cord, globus pallidus, and motor cortex, among other structures, are discussed.

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1 Coordination of movement gaits

During exploration of their environments, terrestrial animals effortlessly generate a variety of coordinated movements which vary in their frequency and patterning to meet momentary task demands. This article continues our description of a family of central pattern generator (CPG) models whose oscillations exhibit the types of frequency changes and gait changes that many humans and animals exhibit as they move at a slower or faster pace. This model extends earlier modeling of these generators that was briefly summarized in Cohen, Grossberg and Pribe (1993). The model is capable of generating parametric behavioral properties of oscillatory movements that have been reported in a number of experimental situations. It elaborates a type of reciprocally inhibitory or opponent processing anatomy that is classical in the motor neurobiology literature (Grillner et al. 1991; Pearson 1993) using neurophysiological voltage-current interactions that have formed a foundation for neurophysiological research since the seminal work of Hodgkin and Huxley (1952).

Oscillatory behaviors place unusual demands on experimental neuroscience because they are typically emergent properties due to interactions among multiple neurons, each experiencing multiple dynamical factors. Correspondingly, the CPGs subserving the oscillatory behaviors simulated here have not been completely "solved" by neurobiological experiments. The present model was derived by using the collective pressure of a large parametric behavioral database, known neurophysiological and anatomical mechanisms, and computational analyses of their emergent network properties. Our goal has been to describe what is perhaps the simplest CPG model that satisfies all these constraints. Once the basic mechanisms are better understood, finer details of neural anatomy and spiking behavior that are consistent with its qualitative behaviors can be incorporated into the model. To this end, the model is used to make a series of neurobiological predictions to guide further experiments concerning the organization of such a CPG and how it can give rise to observed oscillatory behaviors. Along the way, the model sheds light on how simple neural commands generate complex behavioral patterns as emergent properties of network

interactions. Before introducing the model, the experimental background will be reviewed.

It has been known since the beginning of this century that the deafferented low-spinal cat can exhibit muscle rhythms that are characteristic of walking (Brown 1911). Sherrington (1906) claimed that the gaits were generated by a reflex chain. This became a historic debate as to whether afferent sensory signals were a necessary component of pattern generation (e.g., Gray 1950) or not (e.g., von Holst 1954).

Grillner and Zangger (1979) reported that deafferented spinal cats exhibit gaits with different hind-limb phase relationships depending upon the level of electrical stimulation to the spinal cord. It is currently widely held that such oscillations are spinally generated (Grillner et al. 1988; Lundberg 1980; Shik and Orlovsky 1976). Although the existence of intraspinal mechanoreceptors in the lamprey (Grillner and Wallen 1984) casts doubt on some deafferentation experiments, CPGs have been conclusively demonstrated in paralyzed spinal cats. Grillner and Zangger (1979) reported that fictive locomotion in acute spinal curarized cats can be initiated by injection of dopa. Pearson and Rossignol (1991) found that three rhythmic behaviors – stepping, paw shaking, and paw-squeeze response – could be generated by central neural networks deprived of phasic sensory input.

Nonetheless, afferent signals have been established to be important in calibrating the CPG to the animal's environment and to its biomechanical state. Grillner and Rossignol (1978) showed that a bipedally walking decerebrate spinal cat can calibrate its rate of walking to that of a treadmill. These authors showed that sensory input can signal a transition from stance to swing. Afferent signals may also be capable of stimulating activity in a CPG. Phasic input from group Ia afferents can reflexively induce extensor related activity in the cat (Lundberg 1980). Thus, while the existence of CPGs has been established, afferent input plays an important role in generating the final motor output observed in the behaving animal; for reviews, see Delcomyn (1980) and Pearson (1993). Rhythmical modulation of CPG signals is also provided by supraspinal systems, e.g., the cerebellum (Arshavsky et al. 1985). The situation in the insect is less clear; see Pearson (1976b, 1987) and Pearson, Reye, and Robertson (1983).

The present article develops a minimal CPG network, without afferent feedback, that simulates the fundamental behavior observed in spinal CPGs, such as the anti-phase to in-phase transition observed by Grillner and Zangger (1979). In Grossberg, Pribe, and Cohen (1997), the CPG model was tested by simulating behavioral data about human bimanual coordination. The present article simulates biped and quadruped gaits and their transitions. Model properties may help to distinguish the intrinsic behavioral competencies of a CPG from the modulatory influences introduced by afferent signals. Such an analysis should be useful in designing new experiments, especially in light of Pearson's (1993) recent conclusion that "in most motor systems, it is difficult to specify exactly which features of the motor pattern depend upon afferent input."

Much evidence (for reviews, see Edgerton et al. 1976 and Shik and Orlovsky 1976) suggests that quadrupedal and bipedal gaits and gait changes are generated by a spinal CPG in response to a supraspinal control signal. This key control signal is modeled here, and is called an arousal or GO signal. Such a GO signal also plays a key role in neural models of reaching behaviors (Bullock and Grossberg 1988a, 1991), where they are interpreted to arise within the globus pallidus (Horak and Anderson 1984a,b). In this context, the GO signal controls the speed of a reaching movement through time. In the present analysis of oscillatory movements, it is shown how a GO signal can control both the frequency and the phase relationships of human and quadruped gaits. It was already shown in Grossberg, Pribe, and Cohen (1997) how increasing the GO signal could cause a transition from anti-phase to in-phase oscillations, or from in-phase to anti-phase oscillations.

Our model focuses upon interlimb timing; intralimb coordination of flexor-extensor oscillations is not addressed. This separation is supported by data of Pratt and Jordan (1987) which show that the Renshaw cells and Ia inhibitory interneurons are not part of the CPG for locomotion. These authors demonstrated that when strychnine is used to block the inhibitory output of these cell types, there was no interruption in the generation of fictive locomotion. These data do not support models such as the Miller and Scott (1977) model, which require these cell types. Bullock and Grossberg (1991) have proposed an alternative role for Renshaw cells and I_a interneurons as part of a spinal circuit which assures that the trajectories commanded by descending motor commands are not unduly distorted under variable force conditions.

A key issue concerns the manner in which arousal-dependent phase transitions may switch from in-phase to anti-phase oscillations, or vice versa. For example, as discussed in Grossberg, Pribe, and Cohen (1997), Yamanishi et al. (1980) showed that human subjects tend, in a bimanual finger tapping task, to "slip" toward purely in-phase or purely anti-phase from intermediate phase relationships and to exhibit less variability in in-phase and anti-phase than in intermediate phase relationships. Kelso (1981, 1984) showed that coordinated finger movements cannot maintain anti-phase oscillations in a bilateral finger movement task as the required oscillation frequency is increased, but switch to in-phase oscillations at high frequencies. Muybridge (1957) showed that transverse limbs exhibited a pairwise switch from in-phase to anti-phase oscillations when an animal moved from the slower movement of a trot to the faster movement of a pace. Furthermore, Pearson (1976a) observed that there is a stereotypical pattern of gaits which reliably occurs when a cat increases its speed of locomotion. Figure 1 plots these phase characteristics. There are four stereotypical gaits – walk, trot, pace, and gallop – each characterized by different phase relations between the limbs. While the animal might skip from walk to gallop, it never transfers from gallop to walk as its speed of motion increases.

Reading from left to right, each horizontal bar indicates for a single leg the time the foot is off the ground (white sections) and on the ground (black sections). This, somewhat idealized figure is adapted from Pearson (1976a). It shows that, during the gallop, both forelimbs and both hindlimbs have an in-phase relationship. In fact, during a gallop, the cat's fore- and hindlimbs depart slightly from in-phase (Muybridge 1957). Since the in-phase and pure anti-phase relationships occur for most limb pairings across gaits,

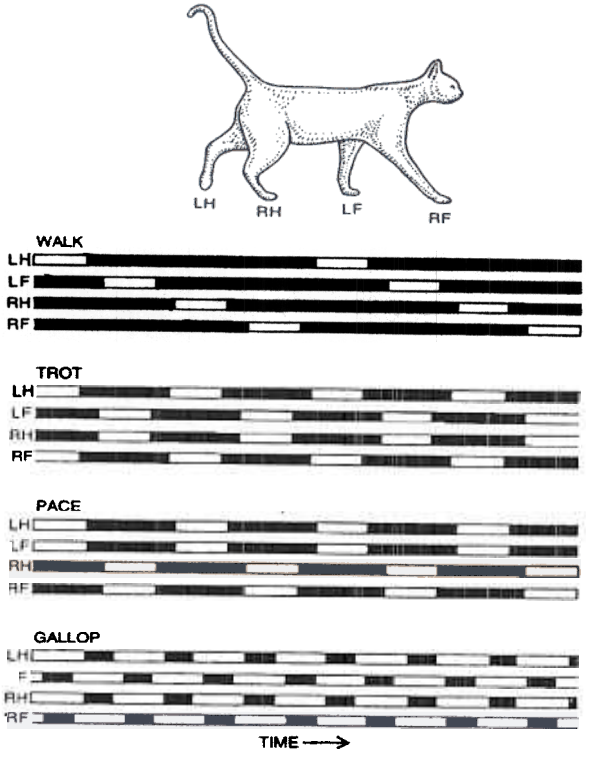

Fig. 1. The stepping patterns of the cat. See text for details

we have focused upon these relationships in our analysis. Simulations not reported here suggest that a small asymmetry in the relative values of E in (2) may be used to induce one limb of an in-phase pair to trail the other slightly, as observed in the gallop. A study of this small effect is a topic for future research.

The CPG model is capable of exhibiting all the frequency-dependent phase transitions that were mentioned above as the GO signal is parametrically increased. The model is defined in terms of a neural circuit from which oscillations are an emergent property. The model variables are the activities, or potentials, of model neuron populations. Various alternative models of locomotion are expressed in terms of operating characteristics of the data, such as the phase angle of the limbs (e.g., Schoner et al. 1990; Yuasa and Ito 1990). Still other models are based on generic, model-independent features of general dynamical systems (e.g., Collins and Stewart 1993). These models permit the application of some general theorems about Hopf bifurcations to study gaits and their transitions (Golubitsky and Stewart 1985). On the other hand, such models do not consider "specific aspects of the intrinsic dynamics of each oscillator or the nature of the coupling between the oscillators" (p. 288) ... "the equations have no particular physiological meaning" (p. 294) ... and some commonly observed gaits "are not found in our modelling analysis ... (and) ... may arise due to detailed aspects of the intrinsic dynamics of the CPG oscillators and/or the nature of the coupling between them" (p. 294) (Collins and Stewart 1993).

The present approach attempts to partially fill this gap. It uses ubiquitously occurring physiological mechanisms, notably model neurons that obey membrane or shunting equations (Hodgkin 1964), which are interconnected in ubiquitously occurring recurrent on-center off-surround networks (Grossberg 1982; Kandel et al. 1991; Kuffler 1953; Ratliff 1965; von Bekesy 1968). The goal is to understand how the anatomies and dynamical parameters of such commonly occurring neural networks could be specialized through evolution for purposes of locomotion. The model CPG oscillators are consequently built out of a minimal number of excitatory and inhibitory model neurons, each of which obeys a membrane equation. The connectivity of the basic model is fixed once and for all. The inhibitory interneurons respond at a slower rate than the excitatory cells. Such slow inhibition is well-known to occur in sensory-motor systems; see, for example, Dudel and Kuffler (1961) and Kaczmarek and Levitan (1987). It has also been proved under rather general conditions that such networks do not undergo oscillations if inhibition operates as quickly as excitation (Cohen and Grossberg 1983; Grossberg 1973, 1980, 1982). The excitatory and inhibitory neurons interact with each other via nonlinear sigmoid signals, another familiar neural constraint; see, for example, Freeman (1975) and Grossberg (1973, 1982). The main result of this article is that, with proper excitatory and inhibitory connections, signals, and relative rates, such a neural design exhibits all the biologically observed gaits as emergent properties when its GO signal is parametrically increased. We therefore call such a model a GO Gait Generator, or G^3 model. Tonic modulation of motor behavior has, in fact, been observed in both vertebrates and invertebrates. For a review, see Harris-Warrick (1988).

Given this basic fact, it then remains to analyse further how these gaits and gait changes can be made as efficiently and flexibly as possible. Some suggestions about how this is achieved are given here. In particular, a G^3 model can generate walk and run gaits in one parameter range as the GO signal increases, and trot, pace, and gallop gaits in a somewhat different parameter range as the GO signal increases. As shown below, the walk and run parameter range is sufficient to provide insight into gaits like the human walk and run, and the elephant amble and walk. For quadrupeds like the cat, which can walk, trot, pace, and gallop, this leaves the problem of how these two parameter ranges can be joined together. Given the available experimental evidence, it is difficult to establish with certainty how this fusion arose during the evolutionary process. Our results make it clear, however, that either two or more copies of the same circuit with slightly different parameters, or one copy of the circuit with parameters that are modulated by the GO signal, could do the job. In particular, given that the basic circuitry can reproduce all four observed gaits, one can begin to see how an adaptive selection process could refine the circuit's basic competence as evolution proceeded.

The most parsimonious solution of this problem is one in which a single circuit exists whose parameters are modulated as the GO signal increases. An analysis of the spontaneously occurring quadruped gait transitions has led us to propose how the GO signal may indeed modulate the functional connectivity of the network in an arousal-dependent way. Such an evolutionary strategy seems to have been discovered long ago, since task-specific modulation of the functional connectivity of neural pattern generators has been experimentally

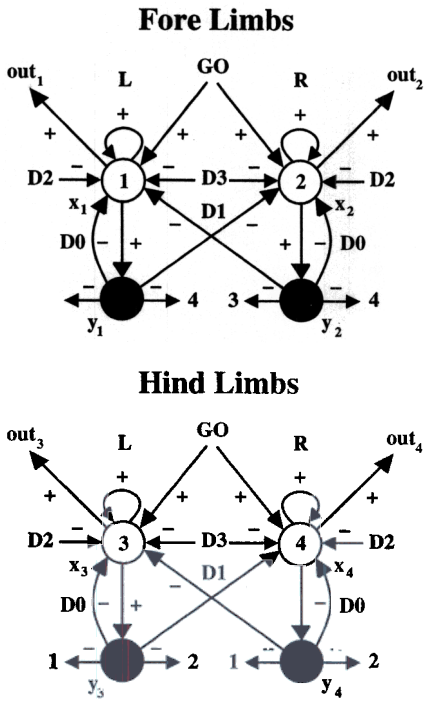

Fig. 2. This figure schematizes the four-channel oscillator for generating phase relationships consistent with all possible quadrupedal gaits by varying arousal level. Inhibitory connections between the fore- and hindlimbs are represented by *arrows* originating at the source of the inhibition and numbered by the label of the node which is the destination. A like-labeled arrow represents the destination of this inhibition. The network has self-inhibition labeled by the parameter $D0$, inhibition between forelimbs and between hindlimbs labeled by $D1$, inhibition between matched forelimbs and hindlimbs labeled by $D2$, and connections between crossed forelimbs and hindlimbs labeled by $D3$

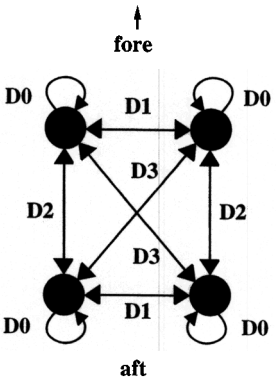

Fig. 3. Key to the reciprocal inhibitory coefficients labels, $D0$, $D1$, $D2$, and $D3$, used in the text

observed in invertebrates; for example, in the stomatogastric ganglion of the crab (Harris-Warrick and Marder 1991; Golowasch and Marder 1992). The present model predicts a prescribed pattern of arousal-dependent inhibitory modulation that permits the naturally occurring quadruped gait transitions, and only these transitions, to be efficiently generated by a single model circuit as its GO signal is parametrically increased within a specified range.

Stafford and Barnwell (1985) have made a related proposal in which the interlimb inhibitory connectivity matrix is changed as a function of a descending tonic signal. In principle, the inhibitory modulation introduced in our model could also be a function, not of the GO signal, but of some other, additional signal. However, any model which relies on a specific descending signal to control gait transitions must be able, in the absence of any modulation of inhibitory synaptic strength, to exhibit the phase transitions observed in the spinal preparation (Grillner and Zangger 1979). Our model has this capability; see Grossberg, Pribe, and Cohen (1997) and the discussion below.

In summary, the approach taken in this research has been to identify several behavioral data sets in different mammal models that could reasonably be argued as fundamental to neural pattern generation, and to identify a single family of CPGs that are built up from commonly occurring neural components and that can generate all of these behaviors. Some of the fine structure shown in these data sets, such as the fine structure of neuronal spikes and bursts, was deemed not to be rate-limiting in this analysis and was not modeled here. Bursting spike patterns and related fine structure can be added using well-studied Hodgkin-Huxley dynamics (Carpenter 1977a,b, 1979, 1981). In this regard, Grillner and his colleagues have shown that they can replicate much of the gross and fine structure of lamprey CPG data (Ekeberg et al. 1991; Wallen et al. 1992). However, in spite of the use of considerably more parameters, they have not yet been able to replicate the gross structure of the Grillner and Zangger data that is demonstrated here. In particular, the model exhibits the phase transitions between in-phase and anti-phase observed by Grillner and Zangger; see also Grossberg, Pribe, and Cohen (1997).

2 The Ellias-Grossberg oscillator

The G^3 model elaborates a family of CPG models that was introduced by Ellias and Grossberg (1975). In these E-G models, the excitatory signals but not the inhibitory signals are coupled to a membrane equation, or shunting, interaction. We found it necessary for both the excitatory and the inhibitory signals to be coupled to shunting membrane processes to generate all the data patterns that are presently simulated by the current CPG model. The G^3 model thus obeys the equations

$$\frac{d}{dt}x_i = -Ax_i + (B - x_i)[f(x_i) + I_i] - (C + x_i)\sum_j D_{ij}g(y_j) \tag{1}$$

and

$$\frac{d}{dt}y_i = E[(1 - y_i)[x_i]^+ - y_i] \tag{2}$$

where

$$[\omega]^+ = \max(\omega, 0) \tag{3}$$

and

$$f(\omega) = \frac{F_1([\omega]^+)^2}{F_2 + ([\omega]^+)^2}, \quad g(\omega) = \frac{G_1([\omega]^+)^2}{G_2 + ([\omega]^+)^2} \tag{4}$$

In Grossberg, Pribe, and Cohen (1997), (1)–(4) are interpreted biophysically in terms of Hodgkin-Huxley dynamics. Here, it suffices to note that the excitatory and inhibitory

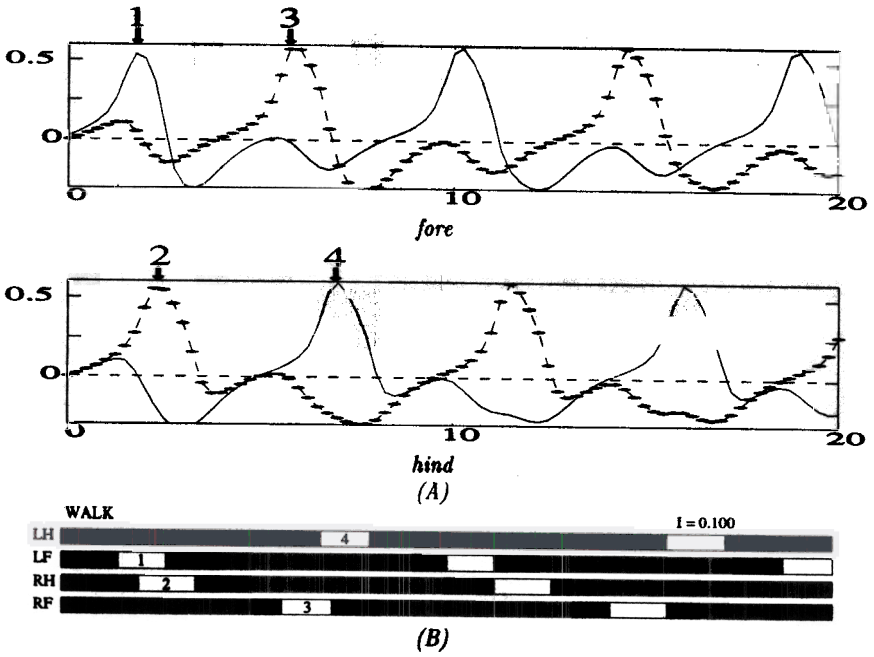

Fig. 4. A An example plot of the oscillator output. The numbered output peaks refer to the correspondingly numbered above-threshold activities in **B**: a diagram of the output shown in the previous figure that has been thresholded at 0.33. The numbered white squares correspond to the numbered peaks in the previous figure. The parameters are A = 1.0, B = 1.1, C = 2.5, $D0$ = 0.8, $D1$ = 0.185, $D2$ aft→fore = 0.0, $D2$ fore→aft = 0.15, $D3$ aft→fore = 0.15, $D3$ fore→aft = 0.0, E = 1.5, F_1 = 9.8, G_1 = 3.9, F_2 = 0.5, G_2 = 0.5. $cordlag$ = 0.0025, $sidelag$ = 0.001, t_{max} = 60.0. The arousal level is I = 0.1

feedback signals $f(x_i)$ and $g(x_j)$, respectively, in (1) are rectified sigmoids as in (4). Each x_i excites only itself, via $f(x_i)$ (recurrent on-center), whereas inhibition may occur via the lateral inhibitory coupling terms $D_{ij}g(y_j)$ (recurrent off-surround). The input terms I_i represent volitional input signals. When only a scalar GO signal perturbs the network, all $I_i = I$.

Oscillations in such a network occur only when the inhibitory interneuronal rate E in (2) is sufficiently small. Indeed, when E is sufficiently large, y_i tracks x_i in (2). Then y_i may be replaced by $[x_i]^+/(1 + [x_i]^+)$ in (1), and the network (1) approaches an equilibrium point under very general conditions on f and g if the coefficients D_{ij} are symmetric (Cohen and Grossberg 1983; Grossberg 1973, 1980; Hirsch 1989). Addition of the shunting term $-y_i[x_i]^+$ in (2), that makes the gain of y_i voltage-dependent, is needed to generate some gait transitions, such as the transition from the walk to the run in bipeds that is simulated in Sect. 6.

3 The four-channel quadruped gait oscillator

A four-channel G^3 oscillator is capable of simulating quadruped gaits and their transitions. Such a four-channel oscillator is designed by appropriately combining two of the two-channel oscillators that were analyzed in Grossberg, Pribe, and Cohen (1997), as in Fig. 2. As in the two-channel oscillator, a single arousal source controls a scalar GO input, I, and reciprocal inhibition occurs between all (x, y) pairs. To simplify notation, the following abbreviations are used in the four-channel parameter lists: the self-inhibitory coefficients D_{ii} are called D0. The reciprocal fore→fore and aft→aft contralateral inhibitory coefficients are all called D1. The fore→aft and aft→fore ipsilateral inhibitory coefficients are called D2. The fore→aft and aft→fore contralateral (transverse) inhibitory coefficients are called D3; see Fig. 3.

The quadruped gaits and gait transitions of the cat – walk, trot, pace, and gallop – were simulated. In order to present the target data, we adopt the display format used by Pearson (1976a). In Pearson's diagrams (see Fig. 1), the movement of each limb is represented by an alternating black and white bar. The time that a limb is on the ground is represented by a black bar. The remainder of the time is represented by a white bar. The outputs of the gait generator are continuous (see, for example, Fig. 4A). To transform this continuous output into Pearson's discrete representation, the output is thresholded and displayed as two distinct levels: white represents suprathreshold output, and black represents subthreshold output. The suprathreshold activity represents the time that the foot is above the ground. The oscillating network activities in Fig. 4A are then displayed as in Fig. 4B. The first four output peaks in Fig. 4A are numbered, and these numbers correspond to the numbers labeling the white bars in Fig. 4B. In this example, a walk is shown (compare Fig. 1). In addition to the walk, trot, pace, and gallop, there is an additional quadrupedal gait called the pronk, wherein all four limbs move together. This gait is not found in the cat. A symmetric choice of parameters can generate a pronk as a four-channel version of the in-phase oscillation discussed in Grossberg, Pribe, and Cohen (1997). It is shown below how to eliminate the pronk while maintaining all the desired cat gaits and transitions.

As in the two-channel case that was studied in Grossberg, Pribe, and Cohen (1997), symmetric initial data, weights, and uniform arousal result in symmetric oscillations or approach an equilibrium point. It is necessary to break this symmetry to understand how asymmetric gaits are generated. Symmetry-breaking can be accomplished by spatial or temporal asymmetries in the arousal signal. It was found that stereotyped temporal lags in the arrival time of the GO signal produced the most reliable results. The time lag with which onset of a new level of GO signal to the hind channel activities, x_3 and x_4, follows onset at the fore channel activities, x_1 and x_2, is called the *hindlag*. The time lag with which the GO signal onset to the right hand channel activities, x_2 and

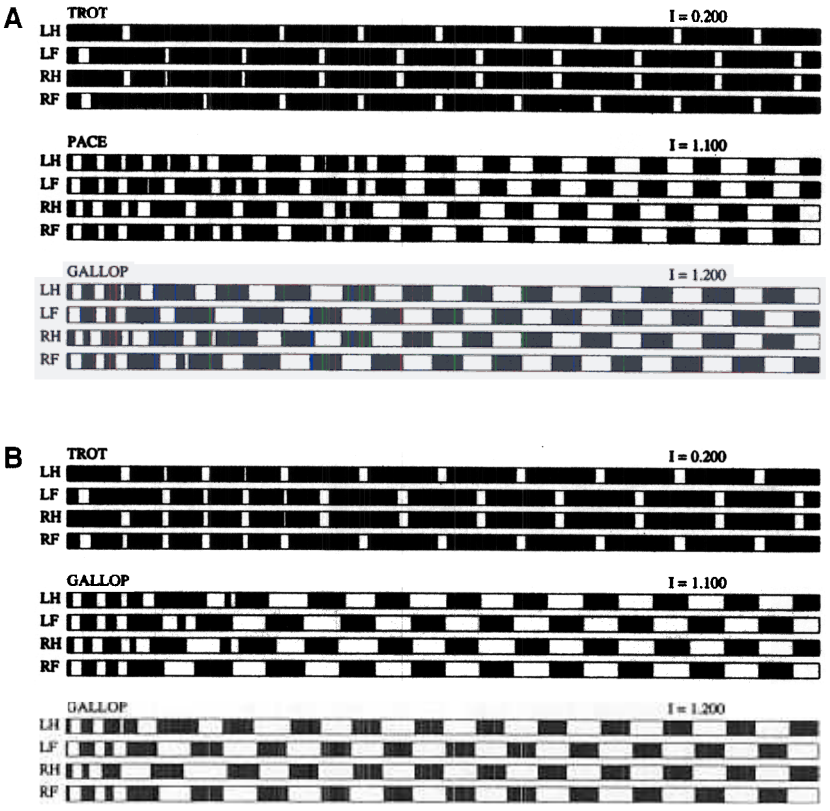

Fig. 5. A Only the arousal was varied to achieve the trot, pace, and gallop. A = 1.0, B = 1.1, C = 2.5, $D0$ = 0.95, $D1$ = 0.255, $D2$ = 0.3, $D3$ = 0.3, E = 1.5, F_1 = 9.8, G_1 = 3.9, F_2 = 0.5, G_2 = 0.5, $cordlag$ = 0.2, $sidelag$ = 0.05, t_{max} = 60.0, and Δt = 0.025. The initial conditions were reset to zero before each gait was sampled. Starting this oscillator with non-zero initial conditions may lead to differing gaits for the same arousal levels. **B** Only the arousal was varied as in the previous figure. The initial conditions were reset to zero for the runs in this simulation. The pace disappears when B is changed from 1.1 to 1.05

x_4, follows onset to the left hand channel activities, x_1 and x_3, is called the *sidelag*. Hence, if the change in arousal, ΔI, arrives at x_1 at time t = 0, then the arousal change arrives at x_2 at time t = sidelag, at x_3 at time t = hindlag, and at x_4 at time t = sidelag + hindlag. This constant set of lags, in the order 1→2→3→4, was sufficient to support all the gaits and gait transitions, despite the fact that different gaits exhibit different symmetries with respect to the four limbs.

4 A simulation of cat gaits and gait transitions

The anatomically symmetric version of the model with temporal asymmetries in the arousal signal is capable, as shown in Fig. 5A, of producing the trot, pace, and gallop in the order shown in Fig. 1. Extensive simulations disclosed, however, that this CPG is sensitive to changes in initial conditions and parameters. Figure 5B illustrates how a parameter change may eliminate one of the gaits in the sequence. Another problem is that, although the phase relationships exhibited in Fig. 1 are also observed in the model output in Fig. 5B, the duty cycles are not. The fraction of the wavelength that activation remains above threshold in Fig. 5 appears too short for the trot and gallop, but too long for the pace, as compared with Fig. 1. Adjusting the threshold used to convert the output of the oscillator to the "binary" form used by Pearson does not improve the model in this regard.

These simulation results showed that the basic GO-modulated opponent CPG has latent within it the types of gaits and gait transitions that have been perfected through evolution. What sort of evolutionary refinements of the CPG could select and stabilize the particular gaits that best fit particular combinations of bodily and environmental constraints?

5 Arousal-dependent modulation of inhibitory gain

A diagnosis of these gaits and gait transitions led to a prediction about how the correct gaits and gait transitions may be consistently and stably generated in a quadruped like the cat. As noted below, an analogous mechanism has been reported in neurobiological experiments on invertebrate CPGs. The proposed mechanism may thus be a variation on an early evolutionary design.

The proposed mechanism takes into account the fact that anatomical asymmetries in the inhibitory coefficients tend to favor one gait over another. The need to generate all possible limb combinations – walk, trot, pace, and gallop – thus recommends a more symmetric choice of coefficients to avoid dominance by a single gait, if these coefficients remain constant through time. Such a choice, however, could create the problem that the correct gaits, and *only* these gaits, may not reliably emerge.

In contrast, one can obtain reliable and rapid gait changes by using asymmetric *arousal-dependent* modulation of the inhibitory coefficients to force gait changes. Such state-dependent modulation converts a single anatomical circuit into different functional circuits that are parameterized by the arousal level. Golowasch and Marder (1992) have reported state-dependent modulation of functional connectivity in the CPG within the stomatogastric ganglion of the crab. The present analysis suggests that a similar strategy may be

Table 1. The values of the modulated inhibitory coefficients for increasing arousal levels, I. See also Fig. 6

	Walk	Trot	Pace	Gallop
	$I \leq .17$	$.17 < I \leq .25$	$.25 < I \leq .35$	$.35 < I$
$D0$	1.0	1.0	1.0	1.0
$D1$	0.3	0.3	0.3	0.55
$D2aft \rightarrow fore$	0.0	0.3	0.55	0.3
$D2fore \rightarrow aft$	0.3	0.3	0.55	0.3
$D3aft \rightarrow fore$	0.3	0.55	0.3	0.3
$D3fore \rightarrow aft$	0.0	0.55	0.3	0.3

deployed in the spinal CPG that controls gait transitions in the cat.

With this addition, the system becomes:

$$\dot{x}_i = -Ax_i + (B - x_i)[f(x_i) + I_i] - (C + x_i)\sum_j D_{ij} h_{ij}(I) g(y_j) \tag{5}$$

and

$$\dot{y}_i = E[(1 - y_i)[x_i]^+ - y_i] \tag{6}$$

Arousal I now performs two functions: it modulates the inhibition and provides the activation that triggers the oscillations. Using state-dependent modulation, stability is realized along with greater flexibility. In particular, other input sources could be used to alter the stereotyped expression of gait transitions. For example, an animal could choose to remain in one gait longer than it otherwise would by using top-down input from the brain to further modulate the inhibition. Term $h_{ij}(I)$ in (5) describes inhibitory modulation by the arousal signal I. Presynaptic modulation via a term $h_{ij}(I_i)$ and postsynaptic modulation by a term $h_{ij}(I_j)$ work equally well to generate quadruped gait transitions in our simulation studies.

Grossberg, Pribe, and Cohen (1997) showed that a primary determinant of phase behavior in the two-channel oscillator is the ratio of the inhibitory coefficients. This fact can be used to guide the choice of inhibitory modulation in the four-channel model: Choose inhibitory modulation for a *fixed* arousal level to move pairs of two-channel oscillators into the phase relationship which would be predicted by that analysis. Thus, to induce a walk, four inhibitory coefficients, the two $D2$ aft→fore and the two $D3$ fore→aft coefficients may be reduced to zero from a base level of 0.3; see Table 1. The $D3$ coefficients may be raised from the base level in order to induce a trot at the chosen arousal level. The $D2$ coefficients may be raised from the base level in order to induce a pace. Raising the $D1$ coefficients while leaving the other coefficients ($D2$ and $D3$) at the base level gives a gallop. Tuning of the arousal dependence in each channel is as shown in Fig. 6. The coefficients of all reciprocal pathways were thus set equal except during the walk.

Figures 7 and 8A present simulations using this arousal-dependent modulation of the inhibitory gain. When a spatial asymmetry in the arousal level is used, it sometimes takes several cycles before the oscillator settles into the desired gait, as shown in Fig. 7. In addition, there is a jump, or pronk, at gait initiation that is not observed in quadrupeds. This problem may be avoided by using a temporal asymmetry in the arrival time of arousal changes, as in Fig. 8A. Since temporal asymmetry implies that different channels may be receiving different arousal levels at the same time,

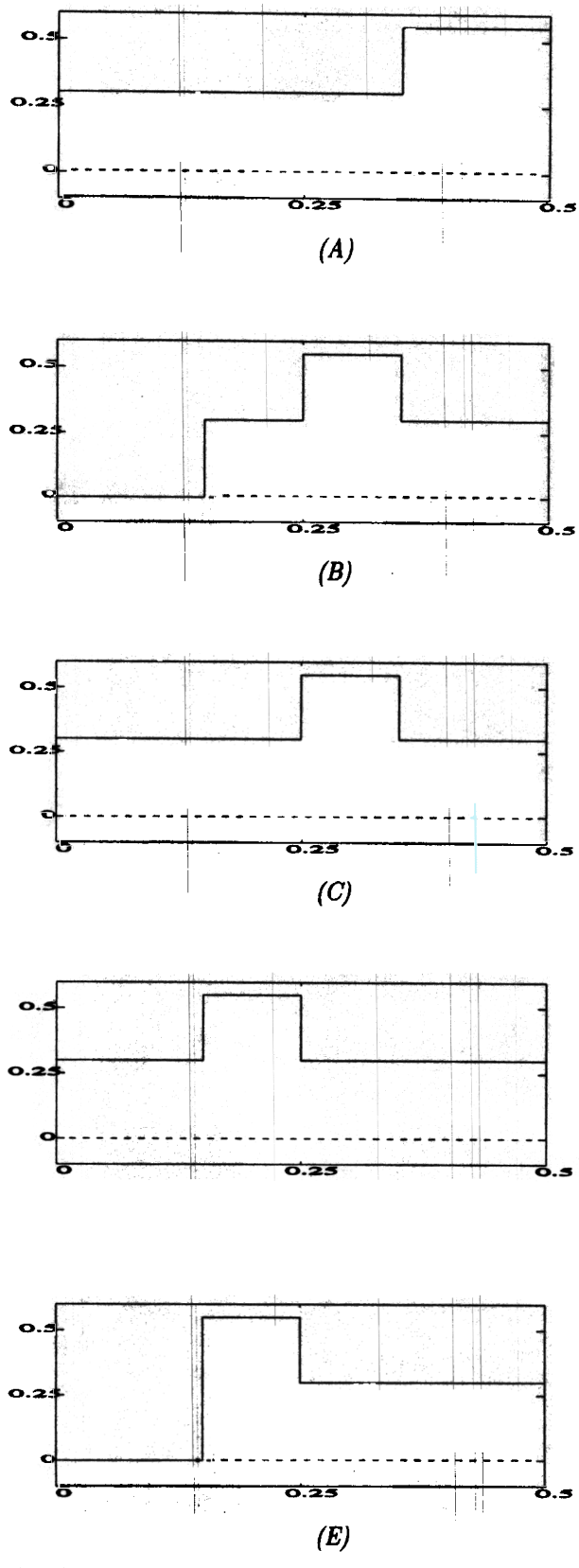

Fig. 6A–E. Plots of the inhibitory coefficient strengths as a function of arousal level I. Appropriate ratios of the inhibitory strengths guide stable switching. Plot **A** shows the strength of $D1$, **B** shows the strength of $D2$ aft→fore, **C** shows the strength of $D2$ fore→aft, **D** shows the strength of $D3$ aft→fore, and **E** shows the strength of $D3$ fore→aft. See Table 1 for the values of inhibitory coefficients as a function of I

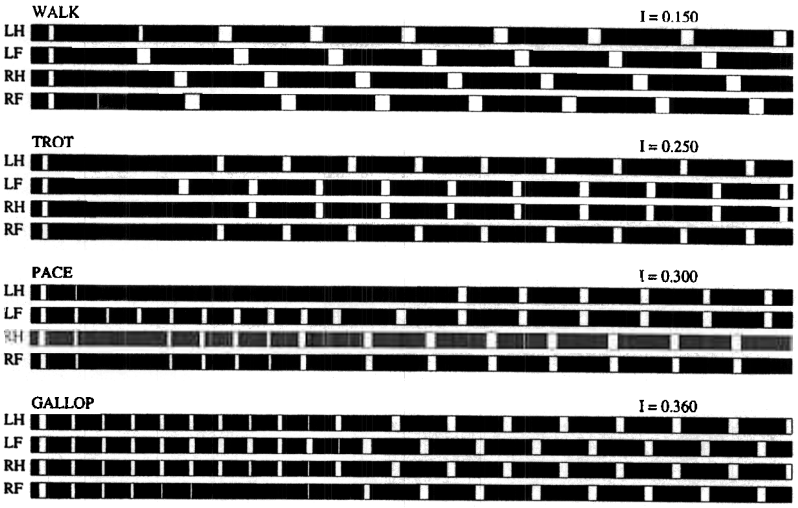

Fig. 7. Arousal-dependent modulation of the inhibitory coefficients with a spatial asymmetry in the arousal signal yields all four gaits. The input $I + \delta$ to x_1 defines the spatial asymmetry. $A = 1.0$, $B = 1.05$, $C = 2.5$; $D0$, $D1$, $D2$, and $D3$ are as specified in Table 1; $E = 1.5$, $F_1 = 9.8$, $G_1 = 3.9$, $F_2 = 0.5$, $G_2 = 0.5$, $cordlag = 0.0$, $sidelag = 0.0$, $\delta = 0.001$, $t_{max} = 30.0$, and $\Delta t = 0.25$. The initial conditions were reset to zero before each new value of I was instituted for clarity and is not a necessary condition of operation of the model

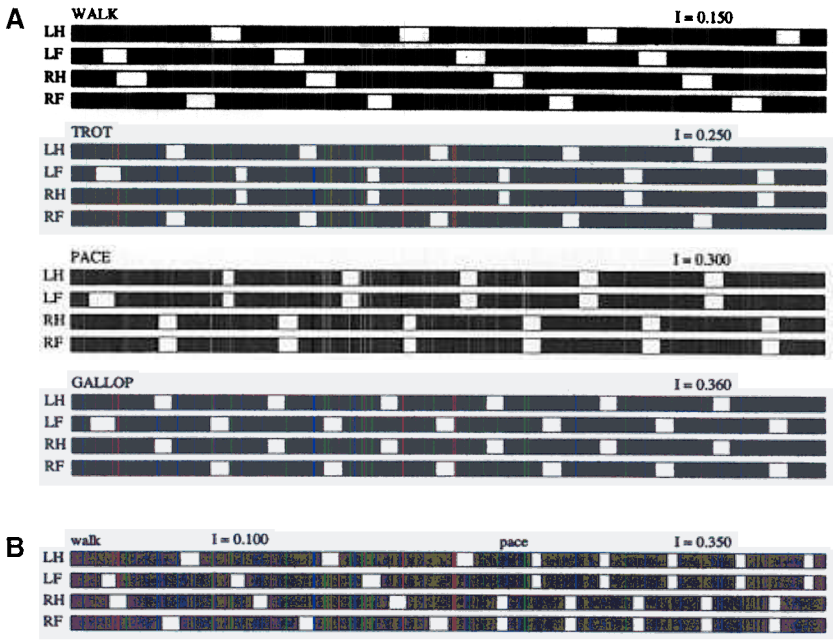

Fig. 8. A Arousal-dependent modulation of the inhibitory coefficients with a temporal asymmetry in the arousal signal yields all four gaits. The temporal asymmetry is a small asynchrony in the arrival time of any change in arousal to the channels. Thus, $cordlag = 0.00025$ and $sidelag = 0.0001$. Parameters A–G_2 are chosen as in Fig. 7 and $\delta = 0.0$, $t_{max} = 30.0$, and $\Delta t = 0.25$. The initial conditions were reset to zero before each new value of I was instituted. Even a small temporal asymmetry can generate fast gait initiation. **B** Initiating a walk from a still position, then generating a transition to a pace. The arousal is instantaneously switched from $I = 0.1$ to $I = 0.35$ at $t = 25.0$. The initial conditions were set to zero at $t = 0.0$. $t_{max} = 50.0$, $\Delta t = 0.25$, and other parameters are as in Fig. 8A

the timing of the inhibitory modulation could be different if the inhibitory modulation depended upon the arousal level of the presynaptic cell, $h_{ij}(I_i)$, the postsynaptic cell, $h_{ij}(I_j)$, or of the command cell, $h_{ij}(I)$. In our simulations, all three choices generated quadruped gait transitions equally well. The plots herein were generated with the command cell timing, $h_{ij}(I)$. A fast gait switch from a walk to a pace is shown in Fig. 8B. A frequency plot of the CPG for the walk, trot, pace, and gallop is shown in Fig. 9. Note the appropriate monotone increase in frequency of oscillations as a function of the GO signal. The model also shows a monotone decrease as arousal increases and successive gaits unfold in the amount of time that the oscillator commands the limb to touch ground (Fig. 8A), as also occurs in vivo (Fig. 1). A more quantitative fit to the data may require linkage of the oscillator to a limb model with afferent and efferent signalling.

6 Gait control of the walk, run, and amble: phase replication

In various quadruped gaits, different relative orderings of limb movements distinguish between gaits. However, the human walk and run gaits both have the same relative limb order. Nor can they be distinguished on the basis of frequency of oscillation, since each gait may exhibit the same frequency: The limbs may oscillate at the same frequency during a fast walk as they do during a slow run.

In addition to the human, the elephant also uses two qualitatively different gaits with the same phase relationship. Where the human uses the walk and the run, the elephant is capable of the amble and the walk. These two gaits in the elephant have the same phase relationship: right-fore, left-hind, left-fore, right-hind. The difference between an amble and a walk in the elephant is readily distinguished by any observer, as is the difference between the walk and the run in a human.

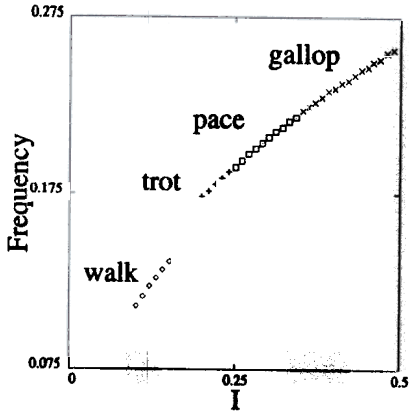

Fig. 9. Frequency plot for the four-channel generator with arousal-dependent inhibitory modulation. The initial conditions were reset at each I increment. The frequencies were sampled at arousal increments of .01. The other parameters are as in Fig. 8

Is there a connection between these biped human gaits and the quadruped elephant gaits? Although humans are bipeds, their arms typically move during normal locomotion, and this movement is coupled to the leg swings. Muybridge (1957) noted that humans use a limb timing pattern similar to the quadruped walk. According to this view, the human does not synchronize the leg and the contralateral arm, as would be the case if human limb timing was analogous to a trot.

In order to understand how two different gaits could exist with the same phase relationships, we exploited the discovery noted in Grossberg, Pribe, and Cohen (1997) that a two-channel G^3 network can generate the same phase relationships with different waveform shapes in different parameter regions. We call this property *phase replication*. The four-channel G^3 network also exhibits two phase replicating regimes that exhibit qualitatively different waveform shapes while maintaining the same relative order of x_i activity. In order to be consistent with the human finger movement and cat leg movement simulations, this hypothesis leads us to interpret the regime occurring at lower arousal levels as a controller for the walk and the regime at the higher arousal levels as a controller for the run. Is this hypothesis consistent with data about walking and running?

Examples of the two different waveforms are shown in Fig. 10. The "walk" oscillations (on the left of the figure) are characterized by sharp peaks that take up a smaller fraction of the cycle than do the more plateau-like oscillations that characterize the "run" (on the right side of the figure). Figures 10 and 11 suggest how it can be that different human gaits cannot be distinguished by relative limb order or even by frequency. The frequency plot for the model walk and run in Fig. 11A shows, as in the human walk and run, that the oscillator can generate overlapping frequency regions. Neither limb order nor frequency can thus be used to distinguish between these two gaits. A measure that can distinguish the gaits is shown in Fig. 11B, namely the fraction of the cycle in which an activity x_i is above threshold. Walks show fractions of cycle above threshold of less than .23, whereas runs are above .31. This property suggests how a limb may have a longer duty cycle – that is, may remain on the ground a larger fraction of the time – during a walk than a run.

These simulations of walking and running gaits and their transition do not require arousal-dependent modulation of inhibitory coefficients. Since only one limb order is required, the bias on the inhibitory coefficients can remain constant across gaits. All that is necessary to switch between the amble and the walk or the walk and the run is an increase in the arousal level. The existence of arousal-dependent inhibitory modulation may thus be expected to occur primarily when symmetry reversals are required across gaits.

7 Discussion

We have described a family of central pattern generator models for the control of the most important quadruped and biped gaits and their transitions. These GO gait generator models are activated by a descending GO signal, or arousal signal, that instantiates the will to act. The internal excitatory and inhibitory nonlinear feedback interactions of the model convert such a descending volitional signal into structured oscillations capable of activating limbs with the orders and frequencies observed during the cat walk-trot-pace-gallop gait transitions, the human walk-run transition, and the elephant amble-walk transition. Rapid switching between gaits with different in-phase and anti-phase properties is facilitated by small, but stereotyped, asymmetries in arousal size and/or timing, supplemented by arousal-dependent modulation of inhibitory signals. Such modulation converts a single anatomical circuit into different functional circuits that are parameterized by the arousal level. Task-specific modulation of functional connectivity in neural pattern generators has been experimentally reported in invertebrates (Golowasch and Marder 1992). We herein predict that modulation of functional connectivity is used in the central pattern generators that control gait transitions in quadrupeds such as the cat.

The use of a GO signal to instantiate the will to act has also played an important role in models of reaching and related skilled arm movements in humans and monkeys (Bullock and Grossberg 1988a,b, 1991; Bullock et al. 1993; Gaudiano and Grossberg 1991; Grossberg et al. 1993). Here the GO signal is interpreted to occur in the global pallidus, based upon neurophysiological data from behaving monkeys (Horak and Anderson 1984a,b). A pathway from the basal ganglia to the spinal cord has also been implicated in the control of spinal movement generators. The G^3 model provides insight into how such a descending pathway can control complex quadruped gaits and their transitions.

In particular, there exists a pathway from globus pallidus (GP) to the pedunculopontine nuclei (PPN) that goes on to the medulla (MED) and finally to the spinal cord (Nauta and Feirtag 1986). This pathway can serve as the means for the expression of the GO signal in the generation of stereotypical gait patterns. Grillner and Zangger (1975) demonstrated that acute mesencephalic cats (precollicular, postmammilary transections) exhibit gait transitions as a function of level of stimulation to the nucleus cuneiforme. Garcia-Rill and Skinner (1987) and Skinner and Garcia-Rill (1990) (also working with precollicular, postmammilary transected cats) re-

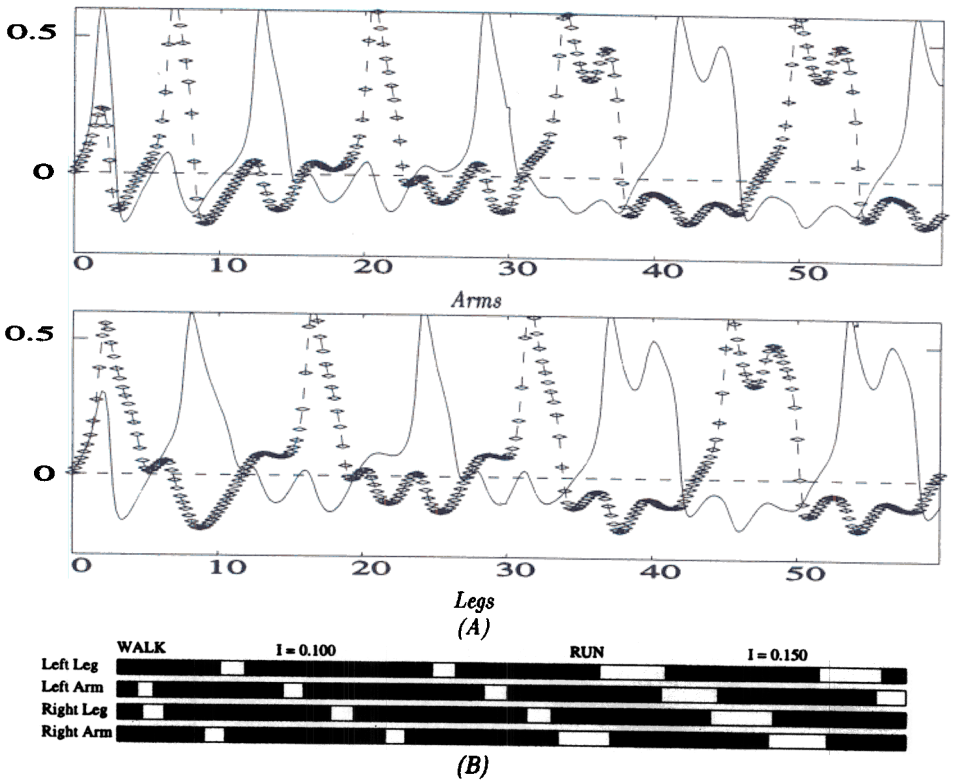

Fig. 10. A A switch from a walk, $I = 0.1$, to a run, $I = 0.15$. Note that the relative phase stays the same, but the shape of the waveform changes dramatically. The other parameters were chosen as in Fig. 4. The arousal increment occurred at $t = 30$, and only the arousal level was changed. **B** A plot of the thresholded output. Note the clean initiation of the walk and the clean transition to the run. The output threshold was .33. The other parameters are as in **A**

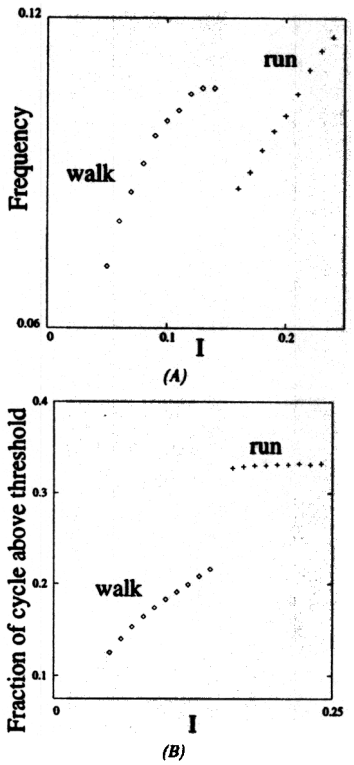

Fig. 11. A The frequencies of the walk and the run. Notice that the walk and the run can have overlapping frequencies for differing arousal levels. Hence, frequency cannot be used to discriminate between the gaits. The frequencies were sampled at arousal increments of .01, and the initial conditions were reset to zero for each sample. Other parameters were as in Fig. 9. **B** The walk and the run can be distinguished quantitatively by the fraction of the cycle that each x_i has suprathreshold activity

ported that the mesencephalic locomotor region (MLR) has as its primary relay to the spinal pattern generator the reticulospinal cells in the medioventral medulla. They also reported that stimulation at either site evokes locomotion. Lai and Siegel (1990) reported stepping-like behavior elicited by consecutive train stimulation to the PPN (which abuts the MLR) and that PPN projects to MED. Garcia-Rill, Skinner, and Fitzgerald (1985) found that by injecting increasing amounts of GABA antagonists into the pedunculopontine nuclei of the cat, gait transitions from a walk to a trot to a gallop could be induced. Skinner and Garcia-Rill (1990) hypothesized a cholinergic/catecholaminergic push-pull process as a neural substrate for generating these and other rhythmic signals. This hypothesis is consistent with the on-center off-surround mechanisms modeled in this paper. Indeed, the ability of these model CPGs to generate both in-phase and anti-phase oscillations suggests that it may be a fruitful basis for modeling other oscillatory processes controlled by distributed neural networks, including those in which either in-phase or anti-phase oscillations are evidence of a pathologic state.

Model properties predict that an animal will tend to always initiate a gait from a standing start in the same way, since a shift in arousal from zero always initiates a new gait in the same phase. The model does not randomly choose a limb to start the gait, but uses a preferred limb to initiate the gait. This property was experimentally observed in a pilot study of the initiation of walking by free-roving dogs; each animal tended to begin moving the same limb each time (Pribe 1991, unpublished manuscript). On the other hand, state-dependent modulation of inhibitory coefficients provides a means whereby top-down signals may be used to supersede the preprogrammed gait of the neural pat-

tern generator. By such means, an animal could continue to trot intentionally at a much higher speed than usual before switching to a pace or a gallop. Arousal-dependent inhibitory modulation is thus a powerful tool for achieving flexible but stable control of neural oscillators in real time.

Stein (1974) derived several properties of interlimb coordination from an analysis of the crayfish swimmeret system. Our work supplements this analysis. Stein noted that the neural network which specifies locomotoric patterns is at once central and distributed. It is central in the sense that the deafferented preparation exhibits the patterns observed in the intact animal. It is distributed in the sense that there is an anatomically distinct rhythmic control center driving each limb. The gait specifying network is, in this view, comprised of three functionally distinct classes of neurons: command, oscillator, and coordinating. Command neurons set the level of excitability of the control centers, but do not directly specify the interlimb phase relationships. Oscillator neurons produce the rhythmic bursts that drive motoneuron discharge. The precise information necessary for interlimb coordination is specified by coordinating neurons. In our CPG model, the command cell output is analogous to the GO signal. The inhibitory potentials governed by (2) and (6) play a dual role: They are a part of the oscillators distributed across the limbs, and they are the coordinating signals specifying the precise interlimb timing.

These results on how neural oscillations may control gaits using their internal feedback dynamics clarify why animals do not always choose a gait with the optimal energy efficiency (McMahon 1984). Explanations of how oscillator parameters are tuned for more efficient gait control may be sought in evolutionary terms, including neural adaptation that may influence the ratios of the modulation coefficients, and thus the arousal levels at which gait switches occur. One factor that may influence such adaptation is the physical dynamics of the muscular and skeletal system, which can also influence gaits both directly and indirectly (Raibert 1990). The physical forces acting on the system during the motion may directly force gait switches. These forces may also have long-term indirect effects by causing differential tissue development, and short-term indirect effects by providing sensory input to the joint or stretch receptors. The interaction of neural pattern generators with such physical constraints requires further study.

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